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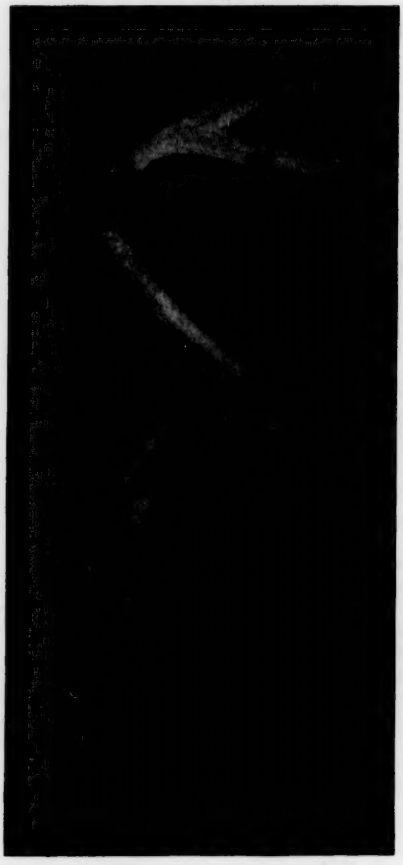

SUPER-SHORT SUPER-PELGER LEG
(SEE PAGE 131)

Test Able and the Genes — Reports on Cotton and Cereals

VOLUME 41 MAY, 1950 NUMBER 5

The American Genetic Association

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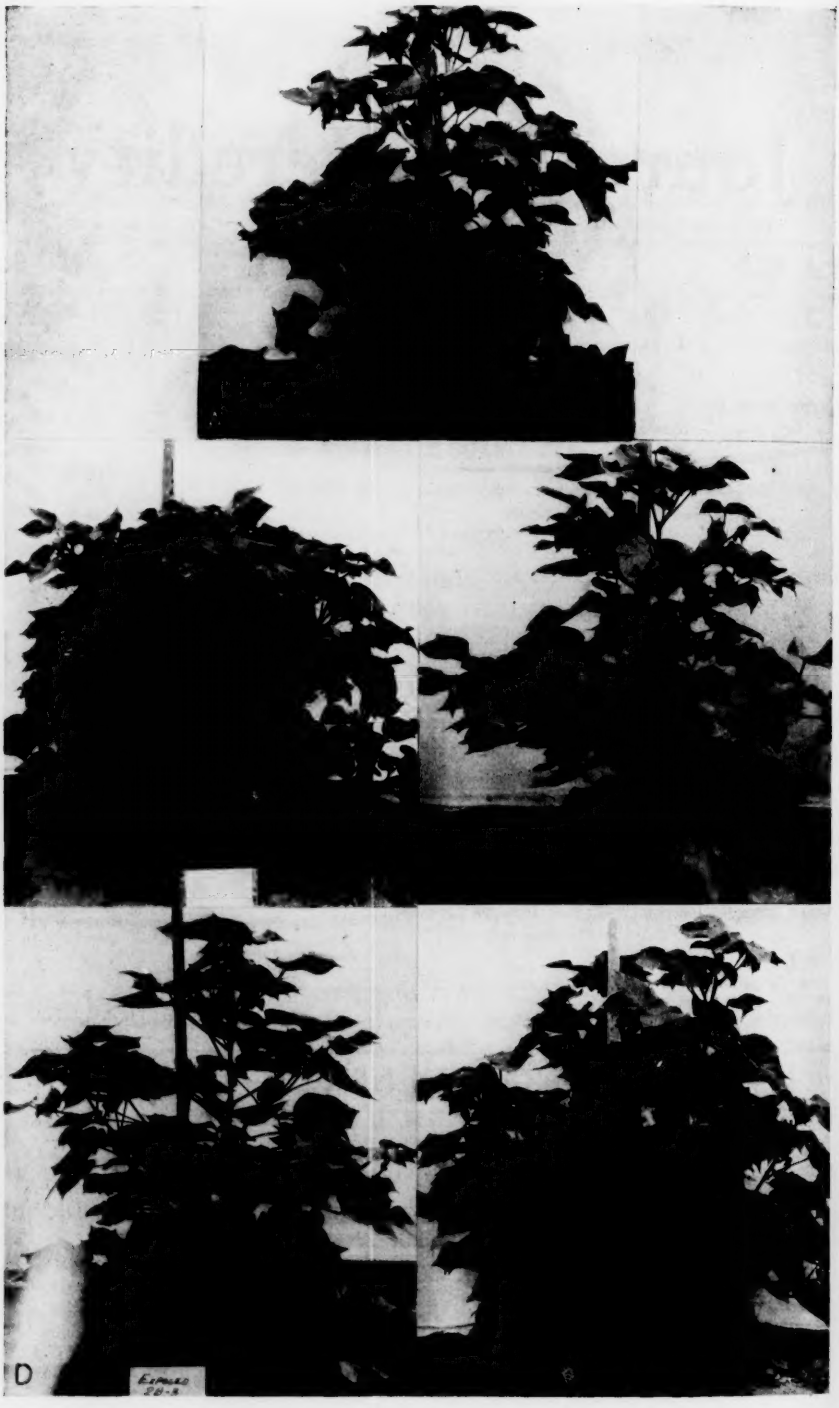

GROWTH DEVIATIONS ASSOCIATED WITH CHROMOSOME CHANGES

Figure 1

A—Normal plant from unexposed seed. *B*—Short compact plant (2B-10) with one chromosome lacking, from seed exposed at Bikini. *C; D; E*—Plants from exposed seed which have chromosome parts broken and rearranged. Part of plant in *C* lacks one chromosome.

COTTON FROM BIKINI

Chromosome Irregularities Found in Plants Grown from Seed Exposed to Gamma Radiation

META S. BROWN*

THE early work of Muller on Drosophila and Stadler on plants first demonstrated that exposure to X-rays can produce translocations, inversions, deletions and other gross as well as minute changes in chromosome structure. Their results have since been amplified by many other workers on many species of plants and animals. The cytological picture of the actual changes in chromosome structure, as demonstrated by Painter, Dobzhansky, McClintock and others, has been correlated with the genetic behavior of the organism. This correlation affords conclusive evidence that the pairing relationships of chromosomes serve as valid criteria for recognizing hereditary chromosome changes. There is now evidence to show that gamma radiation as released during the explosion of an atomic bomb likewise produces such hereditary changes in chromosome structure.

Cytological analyses of the meiotic division stages of pollen mother cells collected from cotton plants grown from exposed seed have revealed irregularities in chromosome pairing which can be attributed only to changes in chromosome structure. Fragmentation, loss of whole chromosomes, translocation of chromosome parts, and possible intrachromosomal changes, were found.

The material used in the present study was obtained through the courtesy of the U. S. Department of Agriculture in cooperation with the Naval Medical Research Section, Joint Task Force One. Seeds of a commercial variety of upland cotton, Acala 911 (*Gossypium hirsutum* L) were exposed at the time of the explosion of the first atomic bomb at Bikini, during Operation Crossroads. Seeds from exposed and unexposed lots of the same variety were planted and grown under identical conditions. Seeds were planted two to a hill, approximately three feet apart. Germination of the exposed seeds was markedly reduced, but no estimate of the reduction in germination due to irradiation can be made since germination of the unexposed lot of seeds was also low. From approximately sixty seeds, fifteen plants, of which one was destroyed, were obtained. Only two hills contained two plants, and no thinning was necessary. In the control lot, seventeen plants remained after thinning to one plant per hill, from approximately 45 hills.

For cytological analyses, buds were collected at intervals of three to fourteen days throughout the growing season, and fixed in a mixture of 30 parts glacial acetic acid to 70 parts 95 percent ethyl alcohol. Acetocarmine smears were made of pollen mother cells undergoing meiotic divisions.

Results

Cytological analyses were made of thirteen of the fourteen plants grown from exposed seed. Of these only one failed to show some disturbance of metaphase pairing. In this plant, *2B-4*, 26 bivalents were found during metaphase of the reduction division, as in normal plants of American cultivated cotton. In some nuclei one bivalent, rarely two, was seen with one chiasma only, but this condition was also found in plants of the unexposed lot, and is not uncommon in any upland cotton. All plants of the control lot from which metaphase stages were studied invariably showed 26 bivalents (Figure *2A*).

In four plants from exposed seed, *2B-2, 9, 7 11*, 26 bivalents were found in a large percentage of nuclei, with two to six univalents in others (Table I).

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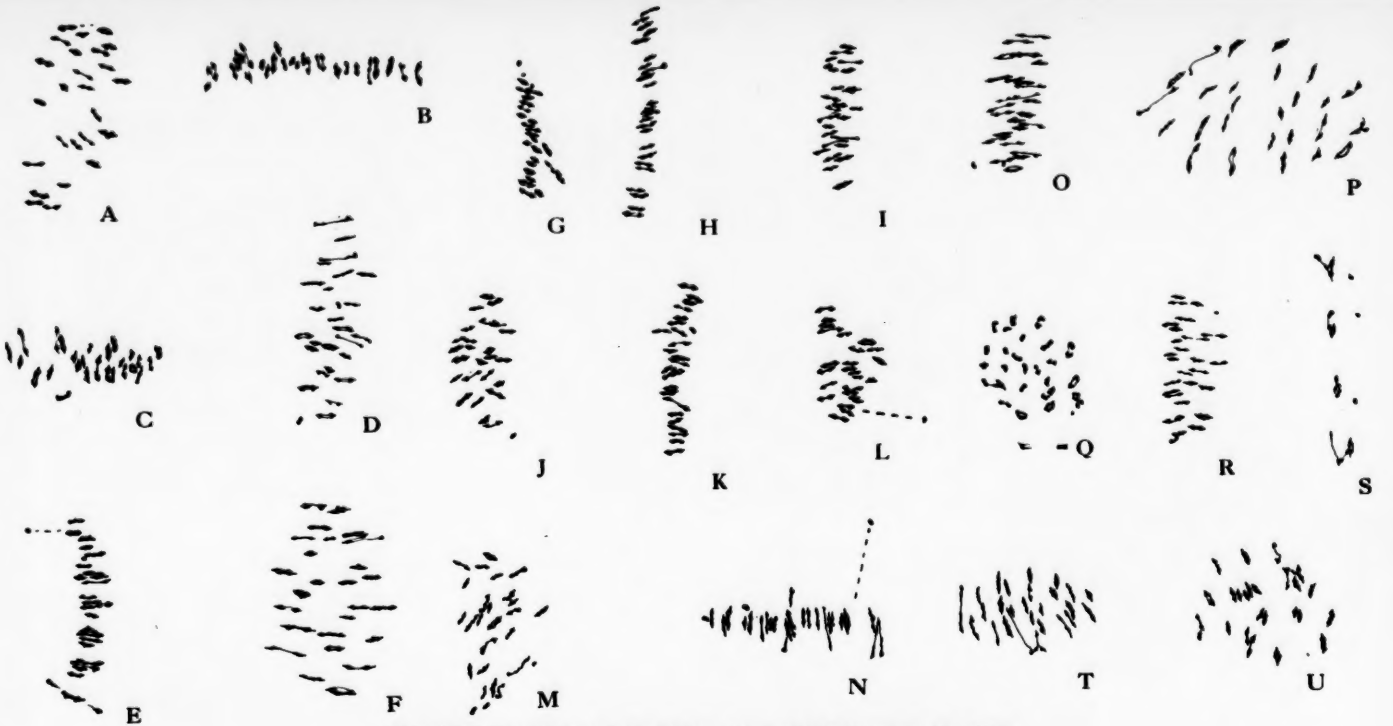


MEIOTIC PAIRING IN NORMAL AND IRRADIATED PLANTS

Figure 2

The preparations shown here are all meiotic metaphase preparations from a normal plant grown from unexposed seed showing 26 bivalent chromosomes (*A*), and from five plants grown from seed treated at Bikini (*B-U*). *B-F* show meiotic anaphase preparations from plant 2B-7. *B* shows 26 bivalents all with two chiasmata. *C*—shows 26 bivalents, of which two are atypical. *D* and *E*—show 25 bivalents, the former with two large univalents, and the latter with two small univalents. *F*—shows 24 bivalents and one quadrivalent. *G-I*—are preparations from meiotic metaphase of plant 2B-6 showing 24 bivalents. *G*—has in addition three univalents, two small and one large; *H* and *I* have one trivalent, formed by the association of a large chromosome with a smaller bivalent. *J-L*—are preparations from 2B-10 showing 25 bivalents and one large univalent. *M* and *N* are from plant 2B-10½ showing 24 bivalents and three univalents, two small, one large (*M*); and 22 bivalents, one large trivalent, and four univalents (*N*). Two bivalents have only one chiasma each. *O-U*—are preparations from plant 2B-1; in addition to the 24 bivalents shown in all but *S*, the following additional configurations are observed: one incomplete trivalent, one univalent, and one fragment (*O*); one incomplete trivalent, and one incomplete bivalent (*P*): one incomplete quadrivalent, and one fragment (*Q* and *R*); one quadrivalent of five parts (*U* and *T*). Note resemblance of quadrivalent to a bivalent plus trivalent. *S*—shows an aberrant type of configuration found in plant 2B-1. Here we find three incomplete quadrivalents with free fragment; one complete quadrivalent of five parts. Figures 2 and 3 were drawn with the aid of camera lucida employing Spencer oil immersion 1.3 (90×) and ocular 10×.

In *2B-7*, pairing was disturbed in at least two pairs of chromosomes as indicated by size differences among the univalents. One pair was large, one small. Chromosome configurations drawn from metaphase nuclei of *2B-7* are shown in Figures *2B* to *2F*. In *2B-9*, pairing was irregular in at least three pairs of chromosomes, two large and one small. In *2B-11*, the chromosomes often remaining unpaired were classified as medium in size.

In three plants, *2B-6, 10* and *10½*, a whole chromosome had been eliminated. At metaphase one chromosome remained unpaired, except in nuclei where it formed part of a trivalent (Table II).

In *2B-6*, the single univalent was a large one; when three univalents were present, two were small and one large (Figure *2B*). When a trivalent was formed in addition to 24 bivalents, a large chromosome was found associated with a smaller bivalent (Figure *2H, I*). In *2B-10*, the size of the univalent indicates that a large chromosome was lost (Figure *2J, 2K, 2L*). In *2B-10½*, where as many as five univalents were found in a nucleus, size differences were apparent, indicating that at least three pairs of homologues were involved, large, medium and small. The missing chromosome was evidently a small one, since when three univalents were present, two were large in size and one small (Figure *2M*); when five univalents were present, two were large, two medium and one small. In some nuclei a large trivalent was present, with or without univalents (Figure *2N*).No quadrivalents were found in any of the three plants in which a chromosome was lacking.

In one plant, *2B-1*, fragmentation occurred in a chromosome with unequal arms. The large portion was invariably associated with a large bivalent and must have been translocated to one of the constituent chromosomes. Attachment was terminal, with little or no loss of chromatin from the bivalent, which always had a chiasma in each arm despite the presence of a large non-homologous fragment. The smaller portion of the broken chromosome, which retained the centromere, frequently did not pair (Figure *2O, Q, R, U*), but passed at random as a univalent to either pole (Table III).

When the free fragment paired with its complete homologue, an asymmetrical, incomplete bivalent was formed (Figure *2P*). When the whole homologue paired with the attached fragment, an incomplete quadrivalent was formed (Figure *2Q, R, S*). When pairing occurred between the whole homologue and both fragments, a complete quadrivalent was formed (Figure *2T, U, S*). Such quadrivalents were always composed of five parts, unlike the typical symmetrical quadrivalents which result from reciprocal translocations.

In four plants, *2B-3, 5, 8* and *12*, quadrivalents, or a trivalent and univalent, were frequently found at metaphase (Table IV).

Although no nuclei were found with more than one quadrivalent in *2B-3*, differences in size of the configuration suggest that chromosomes of three if not four pairs of homologues were altered, since in some nuclei, the quadrivalent was large, in others, definitely small, as com-

TABLE I. Chromosome Pairing in *2N* Plants with Univalents.

Metaphase configurations	Number of pollen mother cells *2B-2*	*2B-7*	*2B-9*	*2B-11*
26 bivalents	17	29	39	53
25 bivalents, 2 univalents	2	9	6	7
24 bivalents, 1 quadrivalent	2	2		
24 bivalents, 1 trivalent, 1 univalent			1	
24 bivalents, 4 univalents		1	1	
23 bivalents, 6 univalents			3	
TOTALS	21	41	50	60

TABLE II. Chromosome Pairing in Monosomic Plants.

Metaphase configurations	Number of pollen mother cells *2B-6*	*2B-10*	*2B-10½*
25 bivalents, 1 univalent	5	8	2
24 bivalents, 1 trivalent	3	1	4
24 bivalents, 3 univalents	1	2	5
23 bivalents, 1 trivalent, 2 univalents	1		4
23 bivalents, 5 univalents			1
22 bivalents, 1 trivalent, 4 univalents			1
TOTALS	10	11	17

TABLE III. Chromosome Pairing in Plant *2B-1*.

Metaphase configurations	Number of pollen mother cells
24 bivalents, 1 quadrivalent	5
24 bivalents, 1 incomplete quadrivalent, 1 fragment	13
24 bivalents, 1 incomplete trivalent, 1 incomplete bivalent	1
24 bivalents, 1 incomplete trivalent, 1 univalent, 1 fragment	2
TOTALS	21

figurations: complete quadrivalent, and one fragment (*Q* and *R*); one quadrivalent of five parts (*U* and *T*). Note resemblance of quadrivalent to a bivalent one incomplete quadrivalent, and one fragment (*Q* and *R*); one quadrivalent of five parts (*U* and *T*). Note resemblance of quadrivalent to a bivalent plus trivalent. *S* shows an aberrant type of configuration found in plant 2B-1. Here we find three incomplete quadrivalents with free fragment; one complete quadrivalent of five parts. Figures 2 and 3 were drawn with the aid of camera lucida employing Spencer oil immersion 1.3 (90×) and ocular 10×.

pared to the rest of the chromosome complex (Figures 3*A, B, C*). Figure 3*D* shows a trivalent and small univalent in *2B-3*. Metaphase configurations from *2B-5* showing a quadrivalent, and a trivalent and univalent, are shown in Figure 3*E* and *F*, respectively. In *2B-12*, where most frequently a trivalent and a univalent were found, the trivalent was small, the univalent very small, suggesting a fragment. In *2B-8* (Table V) two, and even three, multivalent configurations were often found in the same nucleus: trivalents, quadrivalents, pentavalents and a hexavalent (Figure 3*G, H, K, L*). Chromosomes of at least six pairs of homologues must have been affected by the irradiation. When two quadrivalents were found, one was small, one large. A drawing of a metaphase complex showing a small quadrivalent and a large trivalent with correspondingly large univalent is shown in Figure 3*I*. Plant *2B-8* was mosaic for tissue in which one chromosome had been eliminated. Figure 3*G-J* illustrates the *2n* condition, Figure 3*J, K-N*, the *2n*-1 condition. In monosomic tissue, the odd chromosome, whether left as a univalent or associated in a trivalent, was invariably large, indicating that a large chromosome had been lost. Figure 3*K* and *L* shows a large trivalent and small quadrivalent, and Figure 3*M* a large trivalent. Figure 3*N* shows two small and one large univalent as well as a quadrivalent. One bud of *2B-8* contained both *2n* and *2n*-1 tissue; otherwise all other buds showed a total of either 52 or 51 chromosomes, regardless of the degree of pairing.

Four plants, *2B-5, 6, 10* and *12*, and possibly *2B-2*, were mosaic for tissue in which chromosome pairing was always regular. From these plants some buds were collected in which nuclei with 26 bivalents were found. With the exception of *2B-8*, in which all buds analyzed showed changes in chromosome association, any one bud of a plant gave a uniform analysis, some buds showing 26 bivalents, some buds chromosomal irregularities. The data for buds showing normal pairing are not included in the tables. There is some evidence to indicate that in *2B-12* possibly several types of aberrant tissue are present. In one bud 24 bivalents and a quadrivalent were found, in other buds invariably a trivalent and a univalent, as described above.

The analysis of chromosomal changes was made primarily on the basis of metaphase associations at the first meiotic division. A few preparations showing chromosomes at early pachytene and diakinesis were obtained, as well as anaphase, telophase, and second division stages. Some early pachytene figures in *2B-2* and *2B-11* showed unpaired segments. At anaphase univalents were found passing ahead of paired chromosomes to either pole

TABLE IV. Chromosome Pairing in Plants with Quadrivalents or Trivalents and Univalents.

Metaphase configurations	Number of pollen mother cells		
	2B-3	*2B-5*	*2B-12*
26 bivalents	4	23	7
25 bivalents, 2 univalents	1	5	2
24 bivalents, 1 quadrivalent	13	19	2
24 bivalents, 1 trivalent, 1 univalent	4	8	17
23 bivalents, 1 quadrivalent, 2 univalents		1	
23 bivalents, 2 trivalents			1
23 bivalents, 1 trivalent, 3 univalents			1
22 bivalents, 1 quadrivalent, 1 trivalent, 1 univalent		1	
21 bivalents, 2 quadrivalents, 2 univalents		1	
TOTALS	22	58	30

TABLE V. Chromosome Pairing in Plant *2B-8*.

Metaphase configurations in *2N* tissue	Number of pollen mother cells
26 bivalents	2
25 bivalents, 2 univalents	1
24 bivalents, 1 quadrivalent	5
24 bivalents, 1 trivalent, 1 univalent	2
23 bivalents, 1 quadrivalent, 2 univalents	1
22 bivalents, 1 quadrivalent, 1 trivalent, 1 univalent	3
22 bivalents, 2 quadrivalents	3
22 bivalents, 1 pentavalent, 1 trivalent	1
22 bivalents, 2 trivalents, 2 univalents	1
21 bivalents, 1 hexavalent, 1 quadrivalent	1
20 bivalents, 3 quadrivalents	1
TOTAL	21

Metaphase configurations in *2N-1* tissue	Number of pollen mother cells
25 bivalents, 1 univalent	1
24 bivalents, 1 trivalent	4
23 bivalents, 1 quadrivalent, 1 univalent	1
22 bivalents, 1 quadrivalent, 1 trivalent	6
22 bivalents, 1 quadrivalent, 3 univalents	1
21 bivalents, 1 pentavalent, 1 quadrivalent	1
21 bivalents, 1 quadrivalent, 1 trivalent, 2 univalents	2
20 bivalents, 1 pentavalent, 1 quadrivalent, 2 univalents	1
TOTAL	17

CHROMOSOME CONFIGURATIONS FROM BIKINI TREATED SEED

Figure 3

A-B—shows meiotic metaphase from plant 2B-3 all showing 24 bivalents. In addition *A* and *B* show a large quadrivalent. Note difference in type of disjunction; *C* shows a small quadrivalent; *D* shows one trivalent and one univalent. *E* and *F* show metaphases from plant 2B-5 showing 24 bivalents and one small quadrivalent (*E*) and one large trivalent and one large univalent (*F*). *G-N* are metaphase preparations from plant 2B-8 which was a chimera. The sector from which preparations *G-J* were taken had a chromosome complement of $2n = 52$. Preparations from a sector having $2n\text{-}1 = 51$ chromosomes are shown in the remaining figures. Details are as follows: *G*—21 bivalents, one large quadrivalent, one smaller hexavalent; *H-I*—22 bivalents, one large pentavalent, one trivalent; *J*—24 bivalents, one large trivalent, one large univalent. *K-L*—22 bivalents, one small quadrivalent, one large trivalent, one large chromosome missing; *M*—24 bivalents, one large trivalent; *N*—22 bivalents, one large quadrivalent, three univalents, one large, two small.

or left at the equator. A few anaphase bridges, characteristic of inversions, were found in *2B-2, 2B-9* and *2B-1*. Second division stages appeared normal. In most plants the number of microspores per tetrad was four, as in normal plants. In two plants, *2B-3* and *2B-6*, an occasional tetrad with five or six microspores was found.

With few exceptions, the plants grown from exposed seed were equally as vigorous as plants grown from unexposed seed and morphologically indistinguishable from them (Figure 1). All plants were a deep green color with no abnormalities of leaf, bud or flower, with the exception of occasional flowers in which anthers failed to shed pollen. One plant, *2B-12*, showed a slight fasciation in the early seedling stage, but this was soon outgrown and the mature plant exceeded in number of branches and lateral spread all other plants, including the control. Plants *2B-6* and *2B-10*, both mosaic for monosomic tissue, were low, compact, and late to flower, but otherwise vigorous. On one plant, *2B-1*, the initial growth died back before a cytological analysis had been made. It had reached a height of about

eight inches and produced an early, slightly abnormal boll when a lateral bud developed into a branch which became dominant and formed the plant *2B-1* analyzed above. One plant, *2B-10½*, was at first so small that it was not expected to survive and hence left unnumbered. Late in the season it grew rapidly and was analyzed as a monosomic.

Despite frequent collection of buds for cytological analysis, open pollinated bolls were produced on all plants, and selfed bolls were obtained from all except one plant. The four plants with quadrivalents, *2B-3, 5, 8* and *12,* were used in making crosses. Of 53 reciprocal crosses using pollen of one aberrant plant on another, 40 bolls were harvested, which indicates that not only the ovules but the pollen was functional. Of 49 pollinations made on these plants, using an upland stock carrying genetic markers, only four bolls were harvested. These crosses, however, were attempted late in the season after plants had been analyzed, when conditions for boll setting were no longer at an optimum.

Four plants matured early and failed to develop new growth in the fall. The remaining plants resumed active growth after the harvest and of these, seven were transferred to the greenhouse for genetic studies.

Discussion

The chromosome configurations found at meiotic metaphase in the plants grown from exposed seed leave no doubt that the exposure to gamma radiation was effective in producing hereditary chromosome changes. The configurations seen are like those which have been found in material exposed to X-rays or other sources of ionizing radiation. All types of quadrivalent association were found: the zigzag *N*, the closed rectangle (or ring), the angular *U*, the angular 8, and the closed bivalent with attached chain of two chromosomes. In the absence of duplicate chromosomes, as in tetrasomics, or of partially homologous chromosomes, as found in species hybrids, the formation of quadrivalents is best explained on the assumption of translocations, usually reciprocal. Competition between the homologues involved in chromosomal interchange, as well as intra-chromosomal changes which may have occurred, reduces chiasma frequency, and so accounts for many chromosomes remaining unpaired. Loss of whole chromosomes, perhaps through fragmentation, or loss on inactivation of the centromere, is a frequent consequence of irradiation. Intra-chromosomal changes, even large inversions, could not be detected in the above analyses, although some preparations were obtained of prophase nuclei which showed unpaired segments in pachytene chromosomes. Inversions, or other rearrangements may well have been responsible for the occurrence of univalents in plants which showed no other changes.

In cotton chromosomes, which can be studied readily only in the metaphase stage of the first meiotic division, gross structural changes only, such as outlined above, can be detected. By analogy with the results obtained from other types of irradiation, it seems reasonable to assumed that minute rearrangements and deletions as well as gene mutations, may also have been produced.

The detection of visible gene mutations, which tend to be recessive more often than dominant, is best made in stocks which are suitably marked. The lack of any phenotypic changes in the exposed cotton plants indicates that any somatic mutations which may have occurred are not sufficiently dominant to be detected by observation. By growing progeny from selfed seed some mutations may be recovered in a homozygous condition and so become visible. However, the polyploid condition of cultivated cotton means that many loci are present not twice but four times. Hence even the first generation of selfing may not reveal all recessive changes which may have occurred. The suggestion at this stage that such changes may have been produced is conjectural only, and based on analogy with results of other types of irradiation. The proportion of different kinds of chromosome changes is known to vary with the source of irradiation. The above data reveal that gamma radiation can be highly effective in producing hereditary changes in chromosome structure.

The small number of plants obtained from treated seeds, and the high proportion of plants which had chromosome irregularities, suggests that many seeds

which failed to produce plants may have had the embryo killed prior to germination, or in early germination stages.

The dormant state of the embryo at the time of exposure to gamma radiation made possible a high absorption of radiation without killing the embryo in every case. Because of the multicellular state of the embryo, many independent changes undoubtedly occurred. When several types persisted, mosaic structure of the plant resulted. Many cells with an unbalanced chromosome complex were probably soon eliminated, as has been shown in studies of mitotic division following treatment of somatic tissue by X-rays, colchicine, and other substances. Hence the initial incidence of aberrant chromosome structure in the plants grown from exposed seeds may well have been much higher. It is to be noted that the structural changes which were detected in the meiotic divisions of buds are only those which have survived many generations of somatic divisions during growth of the plant. Only those aberrant cells which could compete with normal cells survived.

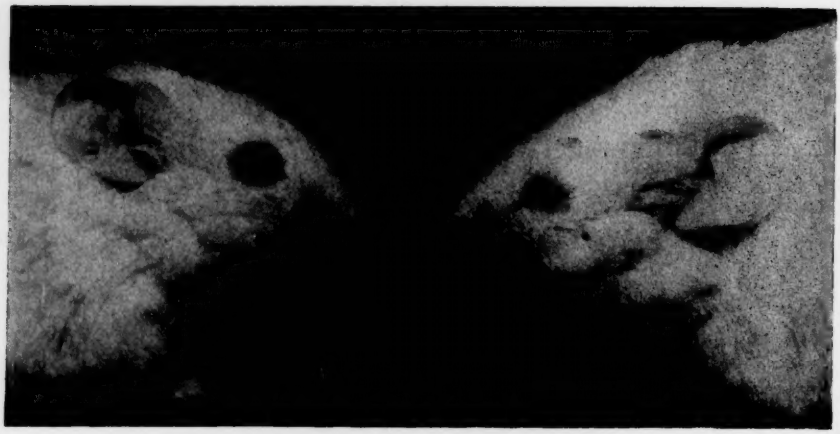
It has been repeatedly demonstrated that translocations and other structural changes in chromosomes have an adverse effect on the fertility of plants and animals. On the basis of chromosome segregation alone, extensive work on translocations in Drosophila and maize by Patterson, Dobzhansky, Beadle, McClintock and others has shown that fertility may be reduced as much as fifty percent. Furthermore, many translocations are lethal when homozygous, thus further reducing the number of offspring. In plants, the post-reduction development of the gametophyte generation makes possible the elimination of much unbalanced germinal material before fertilization, through failure of pollen grains to function. Elimination in the female line is less frequent. In Drosophila, and possibly in higher animals as well, unbalanced gametes of the type which might have been eliminated in plants are viable and may effect fertilization, producing zygotes which die in different stages of development.

From the standpoint of human welfare, proof that ionizing radiation released through the explosion of an atomic bomb can cause breaks in plant chromosomes means than human and animal chromosomes, as well as all others, are also liable to the same kind of injury when irradiated. In addition to the initial lesions which may be produced, there is the possibility of hidden changes in chromosome structure which constitute a potential threat to the fertility of the individual. Complete sterility is of tragic consequence primarily to the individuals concerned. From a broader viewpoint, partial sterility, involving the transmission of altered chromosomes, remains a constantly recurring threat to the production of normal individuals. There remains the possibility that in rare instances superior individuals may be produced. But in experiments with lower animals and plants, such have been greatly in the minority.

From the standpoint of the advancement of scientific knowledge, the demonstration of the induction of chromosome breaks by gamma radiation provides added opportunities for cytogenetic study. Except for satellites on one or two chromosomes, no morphological distinctions have been found on cotton chromosomes aside from length, which is of limited application in a species with 26 pairs of chromosomes. With the production of visible changes in chromosome structure, the affected chromosomes can be studied in relation to different alleles. Individual chromosomes can be morphologically identified, and thereafter associated with specific genetic characters. Information on the linkage relationships of known mutants will be obtainable, and it may be possible to build up balanced stocks carrying desired markers. From structurally altered chromosomes, stocks which carry small duplications or deficiencies can be produced. These will not only provide information on the effects of individual chromosomes, but will be less unbalanced than plants hypo- or hyperploid for whole chromosomes, and in many cases will be more stable.

A NEW MUTATION WITH ASYMMETRICAL EXPRESSION IN THE MOUSE

E. D. Garber and F. C. Hauth, H.M.C., U.S.N.*



MALE LEFT EYE SMALL

Figure 4

The rule that inherited deviations are usually symmetrical is proved by an exception in this strain of mice. Shown in these two views are the normal right eye and the aberrant left eye of the same mouse. Females rarely show the small eye, and it is usually on the left side.

ALTHOUGH inherited variations in paired structures of bilaterally symmetrical animals are usually expressed equally on both sides of the body, exceptions have been reported in the literature, for example, ruby eye[1] in the mouse and microphthalmia[2] in the guinea pig. Another exception has recently been observed in mice in which microphthalmia was not only generally restricted to the left eye but also usually displayed by the male.

Materials and Methods

Microphthalmic mice were noted in the course of establishing inbred lines within the Namru strain developed by this laboratory from albino mice of ABC stock. Brother-sister mating has been rigidly followed to establish the inbred lines. The closed colony of Namru mice has been maintained by pen breeding for the last six years and has been highly uniform as well as free from disease. Three males, each with an aberrant left eye, and one female with both eyes aberrant were found among the litters from a mating between a normal male and three normal females belonging to one inbred line. Additional microphthalmic mice have since been found in certain other inbred lines suggesting that the mutation was present in the colony and had been discovered in the inbreeding program.

The appearance of the aberrant eye was variable. In one mouse, the left eyeball was completely lacking while the right eyeball was normal in size and appearance. In other instances, the affected eyeball was markedly reduced in size (Figure 4). Noticeably opaque eyes were included in a third category. Occasionally, a reduced eyeball was opaque. None of the aberrant eyeballs was examined histologically.

Matings were made according to the following scheme:

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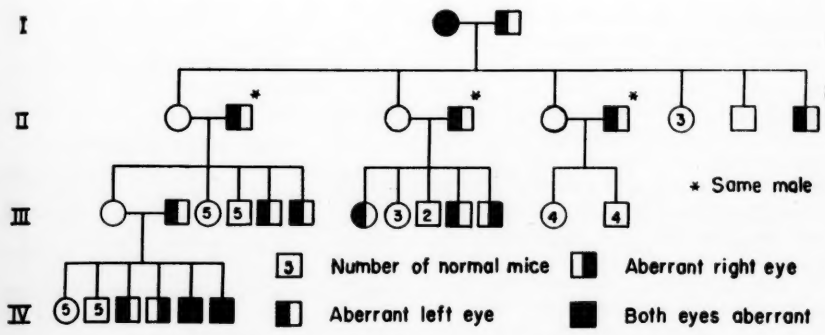

A PEDIGREE OF MICROPHTHALMIA IN THE MOUSE

Figure 5

No simple explanation will account for the peculiar distribution of affected individuals shown here.

Type	Female	Male
1	normal	aberrant left eye
2	both eyes aberrant	normal
3	both eyes aberrant	aberrant left eye
4	normal	normal

Results

Matings between normal females and males with an aberrant left eye did not always yield litters containing microphthalmic mice. In the one cross between a female with both eyes aberrant and a normal male, only one mouse with microphthalmia was observed in two litters. A mating between the same female and a male with an aberrant left eye produced a single microphthalmic mouse. Males with an aberrant left eye were found in litters from crosses between apparently normal parents. Both litters from mating No. 146 included microphthalmic mice. These observations are summarized in Table I. A pedigree of microphthalmia is presented in Figure 5. The data indicate that microphthalmia occurs more frequently on the left side than on the right or on both sides (17/22); and more often in males than in females (19/22). The orginal four microphthalmic mice are included in the comparisons but not in the table. There was no significant deviation from the expected equal number of mice in each sex.[3]

Discussion

The application of simple mendelian formulae assuming complete penetrance to the data in Table I and the pedigree in Figure 5 has not been very successful in clarifying the genetic mechanism underlying the inheritance of microphthalmia. At first, the high incidence of affected males suggested sex-linkage. No manipulation of possible conditions offered a logical basis for this type of inheritance. Stern[4] presents a discussion of two possible explanations for the difference in the sex-incidence of traits. The genetic explanation requires a multifactorial hypothesis in which one factor

TABLE I. A summary of matings between mice either displaying microphthalmia or yielding litters containing microphthalmic mice.

Mating No.	Mating Type*	Progeny ♀	Progeny ♂	Males Aberrant Eye Left	Males Aberrant Eye Right	Males Aberrant Eye Both	Females Aberrant Eye Left	Females Aberrant Eye Right	Females Aberrant Eye Both
26	1	0	0						
27	1	6	3						
28	1	3	2						
29	1	3	4						
76	1	6	7	2					
77	1	4	4	1	1		1		
78	1	4	4						
146	1	5	9	1	1	2			
79	2	11	5	1					
5	3	6	2	1					
20	4	2	2	1					
22	4	6	6	1					
26	4	11	8	1			1		
62	4	7	7	2					
169	4	7	4	1					
Total		81	67	12	2	2	2		

*Mating type refers to the scheme of matings presented in the text.

is sex-linked; the developmental explanation assumes that reactions and interactions induced by the genotype may proceed differently in male and female backgrounds. The appearance of a microphthalmic male in the progeny of a mating (No. 79) between a female with both eyes aberrant and a normal *A* strain male strongly suggests that microphthalmia may be due to a single dominant gene difference. The simplest hypothesis for explaining the inheritance of microphthalmia assumes that the responsible factor is an autosomal dominant with incomplete penetrance and variable expressivity which yields a recognizable phenotype (microphthalmia) more frequently in the male than in the female.

Literature Cited

1. Dunn, L. C. *Proc. Nat. Acad. Sci.* 31: 343-346. 1945.
2. Eaton, O. N. *Jour. Hered.* 28:353-358. 1937.
3. MacDowell, E. C., and E. M. Lord. *Anat. Rec.* 31:143-148. 1925.
4. Stern, Curt. The Principles of Human Genetics. W. H. Freeman and Co. San Francisco. 1949.

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About Time Department

Public Health Division

FEDERAL Security Administrator Oscar B. Ewing issued a warning on March 29, 1950, to shoe stores, their customers and their employees that X-ray machines used for fitting shoes may be dangerous. He called for voluntary control by shoe retailers, and urged communities to set up their own controls if such voluntary measures failed.

In making this warning public, Mr. Ewing said that it was in answer to the many requests received by the Public Health Service for guidance and advice on the use of X-ray shoe-fitting devices in retail stores.

The Public Health Service statement on which Administrator Ewing's warning is based follows:

"Inquiries coming to the Public Health Service about the use of X-ray devices in retail shoe stores originate in the knowledge that such machines are potentially dangerous and that under conditions known to prevail in many stores the dangers far outweigh any beneficial effects the machines may have in the fitting of shoes."

"Use of X-ray fitting machines is almost certain to be harmful when the machines are so constructed as to allow radiation to leak into the surrounding area, or when the machines are out of adjustment, or when the customer's feet are exposed to radiation a number of times in the course of numerous fittings. Children are more likely to be harmed than adults, since rapidly growing tissue is especially susceptible to damage from radiation. If there is radiation leakage, clerks and others who spend considerable periods of time in the immediate area of the machines are endangered."

"A number of community-wide surveys have been made to determine the amount of radiation exposure involved in the fitting of shoes with the aid of X-ray machines under normal store practices. A large percentage of the machines tested in these studies produced far more radiation than is generally accepted as 'safe'. The danger from such machines—to the operators, the customers, and others—is an ever-present one. It is a danger, moreover which increases in direct proportion to the amount of time the machines are in use.

"Since the need for examination by X-ray in the fitting of shoes—except, possibly in cases of malformation—has not been proven, it is the view of the Public Health Service that the use of X-ray shoe-fitting machines should be strictly limited by stores which have such equipment or, if necesasry by local regulation. In cases of malformation, it is assumed that professional advice would be sought."

"Merchants using X-ray shoe-fitting machines should be apprised of the dangers to their customers and employees should make sure, first, that the machines are properly shielded and adjusted, and second, that time limits for examinations are strictly observed. Such voluntary measures failing, community control measures should be instituted."

—*U. S. Security Administration News Release*

EFFECTS OF ATOMIC BOMB RADIATIONS AND X-RAYS ON SEEDS OF CEREALS

A Comparison of the Effects of Ionizing Radiations from the "Test Able" Atomic Bomb and from X-Rays on Seeds of Barley, Wheat and Oats

LUTHER SMITH†

THIS study is part of the cooperative investigation conducted by the Naval Medical Research Section and the U. S. Department of Agriculture to investigate the biological effects of atomic bomb ionizing radiation. A large variety of biological material including animals, seeds, grain insects, bacteria, fungi, etc., were distributed on twenty-two target ships prior to the detonation of the Test ABLE bomb, July 1st, 1946. For additional information regarding the biological aspects of this test, the reader is referred to papers by Draeger and Warren[3] and Randolph *et al.*[4]

Barley seeds were selected as one of the test objects because of their excellent fitness for determining the effects of irradiation on mutation frequency. Young and old seeds were included in order to study the effect of age on sensitivity to irradition. Seeds of durum (tetraploid), common wheat (hexaploid), and common oats (hexaploid) were included in the tests in order to have results from polyploid crop plants for comparison with information obtained from the non-polyploid barley.

Varieties Used and Methods of Handling

Samples of two varieties of barley, 1-year-old seeds of Trebi, and 19-year-old seeds of Moister, were exposed to radiations from the first (aerial) atomic bomb's explosion at Bikini, July 1, 1946. Samples of the seeds were kept as untreated controls and, in addition, some seeds (of Trebi, but not of Moister) were exposed to three dosages of X-rays (4,000*r*, 8,000*r*, and 16,000*r*). Seeds of one variety of durum (Carleton) and one variety of common wheat (Pilot) were handled in the same manner as the seeds of Trebi barley. That is, some seeds were kept as a control; others were exposed to the bomb radiation; and some were X-rayed (4,000*r*, 8,000*r*, and 16,000*r*). Limited observations were also made on the effects of the bomb's

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These studies could not be the result of the efforts of one individual. Broad outlines of the tests were conceived by scientists working with Navy and Army personnel. Specific plans for testing various kinds of biological effects of bomb-created radiations on cereals were made by L. J. Stadler, L. F. Randolph, and M. T. Jenkins. Selection of the varieties of small grains to be used and collection of samples were done by G. A. Wiebe in cooperation with D. W. Robertson and Harland Stevens. X-radiation of seeds for comparison with the bomb-irradiated seeds was performed in the laboratory of L. J. Stadler. Tests on the effects of the radiations on germination and seedling growth were made by R. W. Leukel. Harland Stevens planted the material to be grown to maturity at Sacaton, Arizona. The crop was cared for by R. H. Peebles, who cooperated whole-heartedly in providing facilities for growing and studying the plants.

Macroscopic and cytologic observations on the plants grown at Sacaton were made by Luther Smith, who was also entrusted with the responsibility for determining the mutation frequencies, and with the preparation of this report. Assistance in determining the mutation frequencies in wheat was provided by L. P. V. Johnson. Many thanks for technical help in determining the mutation frequencies are due J. G. Moseman, W. L. Nelson, and C. C. Moh, graduate students in Agronomy at the State College of Washington. As is customary in such a cooperative effort, the writer of this report must accept full responsibility for the accuracy of the observations and for the interpretation of the data presented herewith.

radiations on one variety of oats (Victland).

When the bomb-irradiated seeds (from several points or "stations" varying in distance from the point of explosion of the bomb) were returned to the United States, they were tested for germination in comparison with the control and X-rayed lots. It was observed that the seeds receiving the heaviest irradiation from either the bomb or X-rays were reduced only slightly in germination; therefore, only the more heavily irradiated seeds (bomb and X-rays) were continued through subsequent comparisons. Seeds given 8,000*r* and 16,000*r* units of X-rays were planted at Sacaton, but, on the basis of preliminary observations, only plants from the 16,000*r* treatment were harvested.

The seeds (controls, bomb-irradiated, and X-rayed) were planted in early December 1946 at Sacaton, Arizona, where macroscopic and microscopic observations were made in the spring of 1947 on the plants grown from them. When mature, four widely separated spikes from each plant were harvested and shipped to Pullman, Washington, where tests on mutation frequency were run.

The mutation frequencies were determined in the usual manner (*cf.* Stadler[7]) by planting approximately 30 seeds of each of the four spikes from a plant in separate progeny rows. If a clear-cut seedling mutant character appeared in one or two (rarely two, and practically never in three) of the head rows, it was counted as a mutant. If the character segregated in all of the four rows, the mutation was considered to be of spontaneous origin, prior to the start of the experiment, and was not counted as an induced mutation. Since, almost without exception, four spikes per plant were tested, the mutation frequencies can be expressed on a "per plant" basis by dividing the frequencies by four. However, the four spikes tested would of course not reveal all the mutated sectors in a plant. So the total seedling mutation frequencies would be higher than those reported.

Some effort was made to find mature-plant mutants in X_2* populations; and a number were found in barley and durum wheat, but not in common wheat. However, the observations were on a rather limited scale, and no attempt was made to determine the frequency of mutation for mature-plant characters. It did seem apparent that the frequency of mature-plant-mutants was considerably lower than the frequency of seedling mutants (*cf.* Stadler[7]).

Cytological observations were made on microsporogenesis using the aceto-carmine smear technic[6]. Only one specimen (spike) per plant was examined.

Effects on Barley from Bomb and X-radiations

a. *Germination and Seedling Growth*

Comparisons of the germination of control, bomb-irradiated and X-rayed seeds showed little effect of the radiations on germination—even of the most heavily exposed seeds. However, it was noted that the mottling or "flecking" in the chlorophyll of the first seedling leaves was as heavy in the most severely bomb-irradiated as in the most heavily X-rayed material (16,000*r*). Moreover, it was observed that the mottling seemed more uniform among seedlings from the bomb-irradiated than from the X-rayed seeds.

b. *Macroscopic Observations on Plants Grown from Irradiated Seeds*

Plants grown from control, bomb-irradiated, and X-rayed seeds were gone over carefully about a week before the heading stage in search of evidence that the radiations from the two sources had produced similar or dissimilar effects. During this search three plants from bomb-irradiated seeds were found with distinct, and one with a faint, chimera. Only one distinct chimera was found in plants from X-rayed seeds, and none was found in the controls. The numbers of plants examined were approximately 2,500, 800, and 900 respectively.

c. *Cytological Observations on Plants Grown from Irradiated Seeds*

Cytological observations were limited because the comparison of frequencies of chromosomal aberrations from the two sources of radiation was being carried out with more favorable material—*Zea mays* (*cf.* Anderson[1]; Anderson *et al.*[2], and Randolph *et al.*[4]). However, it seemed desirable to make such a com-

* X_1, X_2 and X_3 designate generations grown from irradiated seed.

parison on a small scale with barley and wheat. The results of these observations are presented in Table I, from which it is evident that the frequency of interchanges resulting from the bomb-irradiation was probably at least as high as the frequency resulting from the 16,000*r* of X-rays.

d. *Comparison of Mutation Frequencies in X_2*

Data on frequencies of mutation are presented in Table II. In Trebi the bomb-irradiated material had a 15-fold increase in mutation frequency over that of the control, as compared with a 10-fold increase in the X-rayed material. The difference would probably have been greater if the number of seedlings per spike (19.3) tested from the bomb-irradiated had equalled the number (25.6) from the X-rayed lots. This difference in population size per spike is considered to have resulted from soil variability rather than to a greater reduction in vigor of plants grown from seeds given the bomb as compared with the X-ray treatment. It so happened that part of the bomb-irradiated material was planted on a distinctly poor spot in the field. This poor eroded spot was near an irrigation ditch and a row of large trees. There was no distinct difference in the average number of seedlings in progenies of spikes with mutants as compared with progenies of spikes without mutants. The spontaneous frequency of mutations in the 19-year-old seeds of Moister barley (0.3 ± 0.15 percent) was similar to the frequency in the one-year-old seeds of Trebi (0.4 ± 0.13 percent). Since these values are based on fairly large numbers of head progenies, the determinations should be reasonably accurate.

As with Trebi, the bomb radiations produced in Moister a 15-fold increase in mutation frequency. Again the number of seedlings per spike was lower from the bomb-irradiated than from the untreated lot. In this case, however, there was no distinct difference in the soil on which the plants were growing. Thus, it seems likely that the bomb radiation reduced the number of progeny per spike in the treated lot.

Effects on Durum and Common Wheats of Bomb- and X-radiations

a. *Germination and Seedling Growth*

As noted earlier with barley, the irradiation seemed to have little effect on germination and seedling growth of the wheats. The wheat seedlings also were similar to those of barley in the presence and distribution of "flecks."

b. *Macroscopic Observations on Plants Grown from Irradiated Seeds*

A surprisingly high frequency of chimeras was observed among the plants from irradiated seeds of durum wheat, but none was found in the common wheat from either the control or irradiated seeds. Eleven plants from the bomb-irradiated seeds of durum had distinct chimeras of "off-color" tissue. There were three plants with such chimeras in the X-rayed material. None was observed in the control populations. The numbers of plants examined were approximately 1,200, 400, and 200, respectively. From the bomb-irradiated, X-rayed, and control lots of common wheat, there were about 1,300, 400, and 200 plants, respectively.

c. *Cytological Observations on Plants Grown from Irradiated Seeds*

Lack of time and other factors made it impossible and undesirable to examine more than a small sample of plants for interchanges and other chromosomal aberrations. The results of these cytological observations, which were limited to specimens taken from the durum wheat, are included in Table I. From the data it is apparent that there was a high frequency of interchanges and chromosomal loss resulting from the irradiation—regardless of source. It is obvious, even from this limited number of observations, that the frequency of interchange was considerably higher in the tetraploid wheat than in the diploid barley. There was a considerably higher frequency of interchanges from the X-rayed than from the bomb-irradi-

TABLE I. Comparison of frequencies of interchanges in plants grown from bomb-irradiated and X-rayed (16,000*r*) dormant seeds of barley and durum wheat.

Stock	Treatment	Spikes with indicated meiotic configurations					
		Normal* No.	R_4 No.	R_6 No.	Other No.	Total No.	Ring of 4 per spike†
Barley (Trebi)	Bomb-irradiated	77	18	3	2[a]	100	0.24
	X-rayed	64	12[b]	0	0	76	0.16
Durum wheat (Carleton)	Bomb-irradiated	16	11	1	2[c]	30	0.57
	X-rayed	12	10	2	7[d]	30	0.83

*"Normal" in the sense that there were 7 (14 of durum) bivalents in the particular sector of the spike sampled by the pollen mother cells under examination.
†Calculated by considering a R_6 as 2 rings of 4, although a R_6 can be derived from 3 breaks (one per chromosome pair) instead of 4—the minimum number of breaks required to account for 2 rings of 4.
[a]These two plants had 2 univalents, one apparently smaller than the other in each case.
[b]One of these spikes had a R_4, 4 bivalents, and 2 univalents, one of which was smaller than the other.
[c]One spike had a R_6 and a R_4; another had a R_4 and 3 univalents plus 10 pairs, a total of 27 chromosomes, one less than the normal number (28).
[d]Four of the 7 spikes had 2 R_4, one of them having also a part of a chromosome missing. The other three had only 27 chromosomes, but each of the three had an interchange in addition to the loss of a chromosome.

ated material, but the number of specimens examined was too small for the difference to be worthy of emphasis.

d. *Comparison of Mutation Frequencies in X_2*

There were two remarkable results in connection with the mutation studies in the tetraploid and hexaploid wheats (Table II). In the first place there were amazingly high, but not strikingly different, frequencies of seedling mutants in the bomb and X-irradiated durum (tetraploid) wheat. Relatively few observations have been made on mutation frequencies in polyploids, but the data of Stadler[8] and general experience, much of it unpublished, have indicated that the mutation frequency in polyploids is very low compared with that obtainable from irradiating similar, non-polyploid species. The reason for this, suggested by Stadler, probably lies in the duplication of genes in polyploids.

In the second place, there was a considerable frequency of mutants in the common wheat—which were, almost without exception, of a particular type, a "banded shrivel." Characteristically, "banded shrivel" seedlings develop (at the late first or second leaf stage) transverse zones of necrotic, brown tissue separated (at least in early stages) by bands of normal green tissue in the leaves. This type of mutant occurred in the barley and durum wheats, but constituted only a relatively small fraction of the total number of mutants. Except for these "banded shrivel" mutants in common wheat, the mutation frequency was very low. The frequencies from the bomb radiation and X-rays were about the same, 0.8 as compared with 1.0 percent.

Effects on Oats from Bomb Radiation

As indicated earlier, the studies on oats were limited to macroscopic observations on germination and seedling growth at Beltsville, and to a search for chimeras at Sacaton. As was noted with the barley and wheat, there was no distinct reduction in germination as a result of the bomb radiation. Likewise, the mottling resulting from the irradiation was comparable to that observed in wheat and barley. No chimeras were observed among the approximately 400 plants grown from bomb-irradiated seeds at Sacaton.

Discussion

One of the most interesting possibilities in all attempts to modify heredity is that an agent will produce new or specific types of changes. For that reason an effort was made to determine whether the types of mutations produced by radiations from the atomic bomb and X-rays were similar. From Table III it is apparent that there were no striking differ-

TABLE II. Comparison of mutation frequencies from untreated, bomb-irradiated, and X-rayed (16,000r) seeds of barley and wheat.

Stock	Treatment	Spikes tested				Progeny per spike		Ratio of normal to mutant seedlings in progenies segregating mutants
		Total	With mutants			Without mutants	With mutants	
		No.	No.	%	σ	Ave. No.	Ave. No.	Average
Barley	Control	2479	11	0.44	.13	24.8	26.0	8.46:1
(Trebi, seeds 1 year old)	Bomb-irradiated	6783	503	7.4	.32	19.3	19.9	3.97:1
	X-rayed	2550	138	5.4	.45	25.6	24.2	4.25:1
Barley	Control	1536	5	0.33	.15	28.5	28.2	3.55:1
(Moister, seeds 19 years old)	Bomb-irradiated	3143	173	5.5	.41	22.9	23.4	4.02:1
Durum wheat (Carleton)	Control	912	0	0.0	0	....	....	....
	Bomb-irradiated	4763	190	4.0	.28	26.8	25.3	4.74:1
	X-rayed	1692	46	2.7	.39	28.7	26.9	5.18:1
Common wheat (Pilot)	Control	1149	0	0	0	....	....	....
	Bomb-irradiated	7324	58	0.8	.10	24.2	24.1	5.62:1
	X-rayed	2400	23	1.0	.20	24.7	25.3	4.52:1

ences in the kinds of mutants obtained from the two sources of radiation.

It was thought to be of some interest to determine the ratio of normal to mutant seedlings in head progenies that were segregating mutants. From Table I it is evident that these ratios were all similar in barley (*ca* 4:1) with the exception of the untreated Trebi (8.5:1). The ratios in the untreated lots were based on relatively few populations but may be suggestive in indicating the period in ontogeny when the mutations occurred. Since all of the radiation-induced mutations resulted from radiation of the dormant seeds, it is to be expected that the segregation ratios might be essentially the same which they were, on the average. On the other hand, spontaneous mutations can presumably occur at any time in the life cycle. Thus, it could be inferred that the 8.5:1 ratio in the untreated Trebi indicated that a considerable proportion of the eleven mutations occurred sometime after germination started. From the same sort of reasoning, again admittedly based on much too small a number of mutants for acceptance as fact, it would seem that the similarity between the ratios of normal to mutant seedlings in untreated and irradiated Moister indicates that most of the five spontaneous mutations may have occurred during the 19-year storage period of the dormant seeds. This problem would justify further study.

It is evident from Table I that the ratios of normal to mutant seedlings in durum and common wheat (4.5 to 5.6:1) were similar but slightly higher than in barley in every case. The reasons for this are not apparent, but the fact that they were so similar indicates that the mutants in these species were monofactorial. X_3 progeny tests also indicate that the mutants in barley, durum and common wheat are monofactorially determined in most cases at least.

It might also be of interest to note that the frequencies of interchanges in spikes of the barley and durum wheat X_1 plants were fairly comparable with the frequencies resulting from a dose of 520*r* units of X-rays applied to the mature pollen of diploid and tetraploid wheat[5]. The above spikes were unselected except that most of them came from later tillers.

However, it should be apparent that in determining the frequencies of interchanges, only one floret of one spike of a plant was examined. This one floret did

TABLE III. Comparison of mutants obtained from bomb-irradiated and X-rayed (16,000*r*) dormant seeds of barley, durum wheat, and common wheat.

	Trebi barley						Moister barley						Durum wheat*				Common wheat*			
Mutant type	Control		Bomb-irradiated		X-rayed		Control		Bomb-irradiated		X-rayed		Bomb-irradiated		X-rayed		Bomb-irradiated		X-rayed	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
White†	2	17	220	44	70	51	1	14	98	57	76	40	16	35	1	2	0	0		
Yellow-green	4	42	87	17	17	12	0	0	30	17	24	13	8	17	0	0	0	0		
Yellow	1	8	48	9	16	12	2	29	11	6	32	17	9	20	2	3	2	9		
Virescent	2	17	41	8	10	7	0	0	9	5	2	1	1	2	1	2	0	0		
Shrivels	0	0	33	7	14	9	0	0	5	3	20	10	6	13	44	76	21	91		
Others	2	15	74	15	12	9	2	29	20	12	36	19	6	13	10	17	0	0		
Total	11		503[a]		138[b]		5[c]		173[d]		190[e]		46[f]		58[g]		23[h]			

*No mutants were found in the untreated control.

†This class, like all the others, includes a wide range of types (though in some cases clearly distinguishable). In other words, there were dozens of kinds of mutants with almost a continuous gradation between types, but they were combined into more or less homogeneous groups for this table.

[a]19 head progenies segregated 2 distinguishable mutant characters, and 4 plants segregated the same or 2 indistinguishable mutants in 2 (in 1 case 3) head progenies.

[b]12 head progenies segregated 2 distinguishable mutant characters, and 1 plant segregated the same or 2 indistinguishable mutants in 2 head progenies.

[c]1 head progeny segregated 2 distinguishable mutant characters.

[d]4 head progenies segregated 2 distinguishable mutant characters, and 8 plants segregated the same or 2 indistinguishable mutants in 2 head progenies.

[e]4 head progenies segregated 2 distinguishable mutant characters, and 4 plants segregated the same or 2 indistinguishable mutants in 2 head progenies.

[f]3 head progenies segregated 2 distinguishable mutant characters, and 4 plants segregated the same or 2 indistinguishable mutants in 2 head progenies.

[g]6 plants segregated the same or 2 indistinguishable mutants in 2 head progenies.

[h]4 plants segregated the same or 2 indistinguishable mutants in 2 head progenies.

not necessarily reveal the chromosomal constitution of the entire spike nor of other spikes on the plant. The number of spikes on the plants of barley probably averaged fifteen (about ten for the durum and common wheats), so it is highly probable that the total frequency of interchanges per plant was several times that represented in Table I.

At the present time there is no explanation for the unexpectedly high frequency of mutations in durum. It may be that the particular variety used has a very old genotype in which evolution has resulted in genic differentiation to a point where the number of duplicate genes is relatively low as compared with other tetraploid types that have been tested. The variety is, however, of relatively recent origin.

Nor is there a ready explanation for the high proportion of mutants in common wheat that were of the "banded shrivel" type. It seems unlikely that these mutants were all the result of induced changes at a single locus. It also seems improbable that all or most of them were the result of the loss of a particular chromosome or part thereof. It is planned to investigate the genetic identity of these mutants.

Conclusion

The results indicate that the atomic bomb-irradiated seeds received about as much radiation as those given 16,000*r* units of X-radiation. It has been estimated that the mean lethal dose of total body irradiation in man is 540*r* and there is experimental evidence that 750*r* is the comparable dose for the mouse. It is evident that the atomic bomb-irradiated seeds received twenty to thirty times the lethal dose for man or mouse.

Summary

The biological effects of radiations from an atomic bomb and from known dosages of X-rays on dormant seeds of barley, durum, and common wheats were determined. The relation of age of barley seeds to susceptibility to radiation effects was also studied. Limited macroscopic observations were also made on plants grown from seeds of oats that had been exposed to atomic bomb ionizing radiation.

The results showed a marked similarity in the effects of radiation from the two sources. The effects studied included: 1) germination, 2) "flecking" or mottling on the first leaves, 3) chimeras, and 4) interchanges in X_1 plants grown from the irradiated seeds; 5) seedling mutation frequencies in X_2, and 6) types of mutants obtained in X_2. From the results, it would appear that the seeds most heavily irradiated during Test Able received a dose of irradiation roughly equivalent to 16,000*r* of X-rays.

There were no clear-cut differences in the types of seedling mutants obtained from the two sources of irradiation nor in the relative proportions of these types.

The frequency of spontaneous mutants (0.3 ± 0.15 percent) from 19-year-old seeds of Moister barley was slightly lower than from one-year-old seeds of Trebi (0.4 ± 0.13 percent). The bomb-induced frequency in the seeds of both lots was 15 times as high as the spontaneous frequency.

Literature Cited

1. Anderson, E. G. *Natl. Acad. Sci. Proc.* 34:387-390. 1948.
2. ————————, A. E. Longley, C. H. Li, and K. L. Retherford. Hereditary effects produced in maize by radiations from the Bikini atomic bomb. I. Studies on seedlings and pollen of the exposed generation. *Genetics* 34:639-646. 1949.
3. Draeger, R. H., and S. Warren. *U. S. Naval Bull.* 47 (2):219-225. 1947.
4. Randolph, L. F., A. E. Longley, and C. H. Li. *Science.* 108:13-15. 1948.
5. Smith, Luther. *Jour. Hered.* 34:130-134. 1943.
6. ————. *Stain Tech.* 22:17-31. 1947.
7. Stadler, L. J. *Jour. Hered.* 21:3-19. 1928.
8. ————. *Natl. Acad. Sci. Proc.* 15: 876-881. 1929.

THE PELGER-ANOMALY IN MAN AND RABBIT

A Mendelian Character of the Nuclei of the Leucocytes

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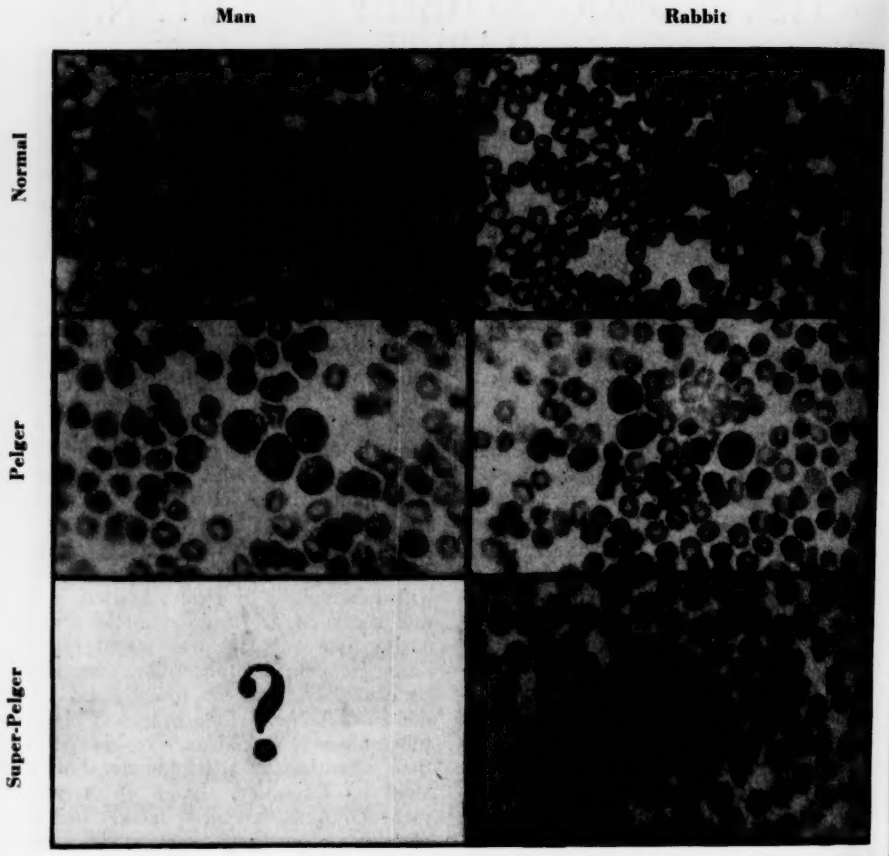
THE polymorphism of the nuclei of the leucocytes, especially of their most frequent type, the neutrophiles, is the typical character of these blood cells. The immature forms of the neutrophiles, the myelocytes, have nuclei which are still unsegmented. As these cells grow to maturity the nucleus stretches and divides into two, three, four, five, and more segments, separated by bridges and at last by delicate filaments of chromatin. In this way a very varied nuclear picture develops, which in the final result leads to a big enlargement of the nuclear surface compared with the original single round nucleus.

In normal blood the different degrees of segmentation are in a fixed proportion. Thus we find in man about 4 percent neutrophiles with unsegmented nuclei (single), 21 percent with two segments, 48 percent threefold, 23 percent fourfold, 4 percent fivefold and multiple segmented nuclei. This proportion shifts in favor of the less segmented forms during infections and inflammations. We then speak of a "shift to the left." In the case of other diseases, such as pernicious anemia, we see an increase of the multiple segmented nuclei—a "shift to the right."

The Pelger-Anomaly in Man

In 1928 the Dutch tuberculosis specialist, K. Pelger,[9] recognized a peculiarity of the leucocyte nuclei differing from the normal in two of his patients. These nuclei persisted, unsegmented when mature or strangulated only once, so that there were two segments. Very seldom he saw three segments, and never were there nuclei with multiple segments. Here a very strong "shift to the left" existed, and it was permanent, while other such shifts are only transitory. As a result of further investigations by G. J. Huët[1] and others, this arrest of the segmentation of the nucleus proved to be a dominant autosomal Mendelian character. Furthermore the fine structure of the nuclei is also altered. The chromatin is coarse, more lumpy than normal, richly interspersed with nucleoli: it stains intensely with basic dyes. Perhaps the arrest of the segmentation is due to the strong pyknosis of the chromatin. Besides the nuclei of the neutrophiles those of the eosinophiles are altered in a similar way.

At first the Pelger-anomaly, as it is now called, was spuriously associated with tuberculosis by Pelger himself, and was regarded as of unfavorable prognosis. The anomaly was found especially frequent in tuberculous families because they had been investigated for the anomaly more than others. Chiefly tuberculosis specialists concentrated their attention on the anomaly shortly after its discovery. Later on it was regarded as a harmless blood variant without any clinical importance: as merely a "sport of nature." But it is not very likely that this obvious alteration of the main character of the neutrophiles should be without any consequences. On the strength of phagocytose experiments Leitner and Gugelot[4] stated that the mature Pelger-cells, indeed, have not been damaged in their functions, but that the immature Pelger-cells do not act as phagocytes as do normal immature cells. From this the two authors came to the following conclusion: if, in case of a severe infection, the peripheral blood is full of immature cells, and these immature cells must act as the defence of the body, a Pelger individual might more easily succumb than an individual with normal neutrophiles.



BLOOD-PICTURE IN MAN AND RABBIT

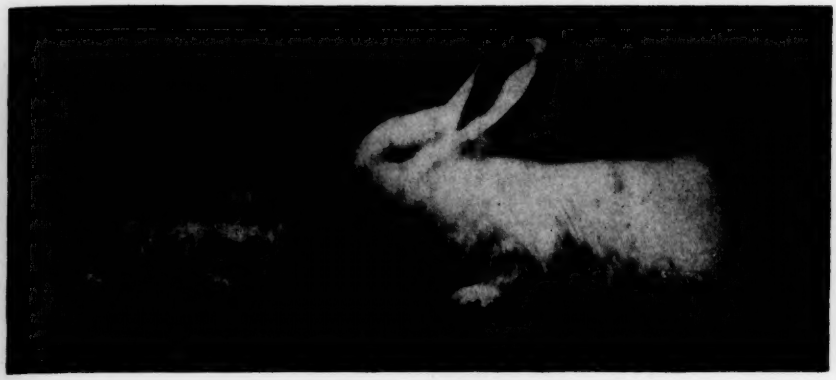
Figure 6

Upper row: Normal blood picture of man and rabbit. Some neutrophiles are shown having multiple segmented nuclei. *Middle row:* Blood picture of man and rabbit heterozygous for the Pelger gene. The nuclei of the neutrophiles are unsegmented or have two segments only. *Lower row:* Blood picture of a rabbit homozygous for the Pelger gene. The nuclei of the neutrophiles are entirely round without any segmentation. The homozygous condition of Pelger men is unknown so far.

For a time the Pelger-anomaly was supposed to be a very rare blood variant. Up to 1940 when I started with my own investigations, 32 families with 210 Pelger individuals had been described, 10 of them each in Holland and Germany, 6 in Switzerland, one family each in Czechoslovakia, France and the U.S.A., and also three cases in people of the mongoloid races, in Chinese, Japanese, and in Malay[5]. So far as I have been able to ascertain, no case has been described in Great Britain up to now; and only two cases[10, 11] have been reported from America.

The Pelger-Anomaly in Rabbit

The investigations on the Pelger-anomaly came into a new phase, when in 1938 the Swiss haematologist E. Undritz[12] showed that the same alteration of the blood picture existed as



PELGER LITTERMATES FOUR WEEKS OLD

Figure 7

The homozygous super-Pelger on the left weighed 180 grams at four weeks; the heterozygous Pelger weighed 710 grams. The super-Pelger had microphthalmia of both eyes as well as a very defective skeleton. (See Figure 9.)

a hereditary character in the rabbit, an animal eminently suitable for experiments. Among 400 rabbits examined from the area of Bâle Dr. Undritz found a buck with a typical Pelger-blood picture. When mated with a normal female, this rabbit produced half Pelger and half normal offspring—both sexes being affected. Dr. Undritz was kind enough to let me have some of these Pelger-rabbits for further investigations.

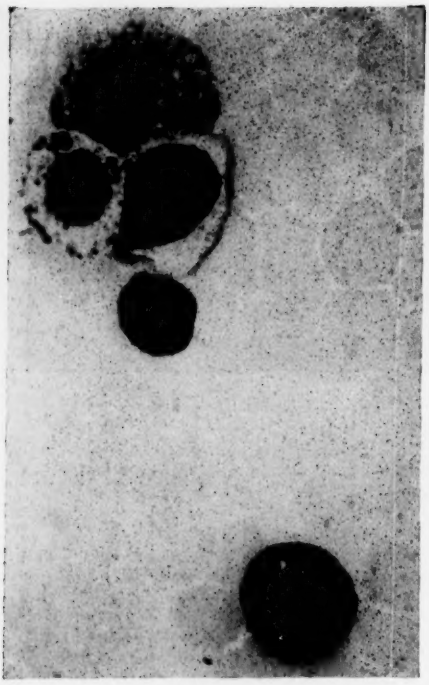
The morphological picture of the Pelger-anomaly of the rabbit corresponds completely to that of man (Figure 6). The nuclear segmentation of the neutrophiles, called pseudo-eosinophiles because of their stronger granulations in the rabbit, is seldom of more than two segments. In both man and animals the tendency towards the formation of a certain percentage of nuclei with three segments is restricted to single individuals or families. Evidently this slight "shift to the right" depends upon the genotype. It is always possible to classify a rabbit with certainty as either normal or abnormal. Man and rabbit also agree in the strong pyknosis of the chromatin of the neutrophiles, and all the other forms of the granulocytes have to undergo a similar change.

Crossing Dr. Undritz's Pelger-rabbits with non-Pelgers we obtained, as expected, a 1:1 ratio. The dominant inheritance of the Pelger-gene with 100 percent penetrance has always been manifested in all our crosses, as shown in Table I. The prenatal mortality of the Pelgers does not exceed that of the non-Pelgers.

We get an unexpected result, however, when two such Pelger-rabbits are mated. Instead of 3:1, the ratio of Pelgers to non-Pelgers among the progeny is 2:1. On an average these F_2 litters were smaller than those of the backcross generation. Besides, all F_2 Pelgers examined proved to be heterozygous, so that we are forced to the conclusion that in a double dose the Pelger gene has a lethal effect and that the homozygotes perish as foetuses. Fortunately, this lethality is not absolute. Some homozygous Pelgers live until parturition or even longer. And the study of these homozygotes, or super-Pelgers, as I call them, has become very informative for the understanding of the Pelger-anomaly.

Table II gives the totals observed in eight years of experiment. Of 701 progeny examined, 439 were Pelgers and 223 non-Pelgers, a nearly perfect 2:1 ratio. In addition there were 39 super-Pelgers, where a number equal to the non-Pelgers (223) would be expected. This means only 17.7% of expectation. Of these 39 super-Pelgers 27 perished during parturition or shortly thereafter. Of the 12 survivors all but two died within the first months of their lives. These two exceptional rabbits, a female and a male, we succeeded in rearing to maturity. The buck could be used for breeding, and his homozygosity for the Pelger-gene could be proved in this way experimentally.

The blood picture of the super-Pelgers is of special interest. In the homozygous Pelger a complete uniformity of the nuclei has been substituted for the polymorphism of the normal granulocyte nuclei, which is already strongly restricted in the heterozygous Pelger. The nuclei look entirely round, there is no stretching, and we do not see the slightest segmentation. This is a shape of the leucocyte nuclei which has never before been observed in mammals. The round shape of the nucleus is typical for all leucocytes of the super-Pel-



LEUCOCYTES OF HOMOZYGOUS RABBIT

Figure 8

Blood picture of a homozygous Pelger-rabbit showing five different types of leucocytes. From the top; eosinophilic, monocytic, pseudoeosinophilic, lymphocytic, and basophilic. All nuclei are perfectly unsegmented and round. The chromatin is strongly pyknotic.

ger. Figure 8 shows a photomicrograph with the different leucocyte types; eosinophilic, monocytic, pseudoeosinophilic, lymphocytic, and basophilic; it is by accident that they are all in one field of vision. All these round nuclei have a strong pyknotic chromatin. The blood pictures of all the homozygous Pelgers examined are the same. No deviation "to the right" has ever been observed.

Homozygous Pelgers

While heterozygous Pelgers and non-Pelgers can be distinguished only by means of blood tests, we can recognize at birth a homozygous Pelger without any difficulty. Its birth-weight and appearance are different. In its development it lags behind the other rabbits of the same litter, and this difference between the homozygote and its littermates increases after birth (Figure 7).

The much disfigured limbs are an even more distinctive and more reliable character of the super-Pelger at birth. In Figure 9 a new-born homozygous Pelger rabbit is shown beside a normal newborn one, both stained by the Alizarin method. The long bones are extremely shortened and curved; and radius and ulna, as well as tibia and fibula are twisted around each other. In older animals radius and ulna are often fused, and the epiphyses are thickened (Figure 10). The bone anomalies naturally greatly reduce the functional fitness of the extremities. The fore limbs are held projecting laterally from the body. They remind one in form and posture of a fin-supporting phocomelia. With these deformed appendages the rabbit is hardly able to move by "paddling." In growing super-Pelgers further symptoms develop; formation of a scurf around the muzzle and nose, a strong flow of saliva and an extreme emaciation. In one case microphthalmia was observed.

Recently my co-worker, Hans Klein,[2] has carried through a detailed pathologic-anatomical and histological examination of the super-Pelger. The most remarkable deformities concern the skeletal system. The histological picture corresponds in many ways to that of the homozygous lethal Creeper fowl described by Landauer,[3] namely hyperplasia of the diaphyses, a chondrodystrophy. Extremely shortened long bones of the extremitities are always found, and in most of the cases the ribs also are affected. In contrast to normal ribs, the ribs of the super-Pelgers are short and clumsy with ends thickened like mushrooms; histologically they are characterized by all the symptoms of chondrodystrophy. The thickening and shortening of the ribs leads to a fixation of the thorax and this is taken to be the cause of death of most of the super-Pelgers immediately after birth. The process of the expansion of the lungs is interrupted, and breathing is only possible by contraction of the diaphragm. Thus an em-

NORMAL AND SUPER-PELGER SKELETONS
Figure 9

New-born rabbits, non-Pelger and super-Pelger, stained with alizarin. The long bones of the extremities of the super-Pelger are much shorter than the bones of the normal rabbit. The ribs are shortened and the ends of the bones have mushroomlike thickenings.

physema of the lungs develops in the newborn young, making them gasp for air and leading to an early asphyxiation. Only a few animals, whose ribs have not been affected or which have only suffered slight deformations by chondrodystrophy escape death.

The time of birth brings the second crisis of development for the super-Pelgers. Thus far it is unknown at which point in embryonic development the first crisis becomes effective, which kills the large majority of the super-Pelgers. The investigation of this problem is more difficult with rabbits than with fowl, whose development takes place outside the mother's body. Nor is it yet possible to say which factors are responsible for the fact that a small percentage of the super-Pelgers survive the first critical period. It is remarkable, however, that the surviving super-Pelgers are not distributed by chance within the F_2 litters. Certain does produce them again and again, while other does do not bring to birth any super-Pelgers. As mentioned above, no more than about 17.7 percent of the theoretically expected super-Pelgers in F_2 were observed in the total material. If we take into consideration the does with super-Pelger offspring only, we get up to 37 percent surviving super-Pelgers among the progeny. Here, too, a problem is still awaiting solution.

The Frequency of the Pelger-Gene in Man

Up to now human homozygous Pelgers have not been observed. The far-reaching parallelism of the symptoms, however, leaves little doubt that the blood picture of the homozygous Pelger in man would be similar to that of the homozygous Pelger-rabbit, provided that in man the Pelger-gene in a double dose has not always a lethal effect during embryonic development. Also if some human homozygotes should survive birth we might expect phocomelia and chondrodystrophy. Examination of some individuals with these or similar deformities of the skeleton showed a normal blood picture. But this result does not mean very much, as these deformities may be genetically very different, and homozygous Pelgers are bound to be very rare. How rare they would be depends on the frequency of the Pelger-gene in man.

Up to this date there were only very insufficient estimates of the frequency of the Pelger-gene in man. They varied between 10 and 3 per ten thousand. To get adequate data to estimate the frequency and the mutability of the gene, we started with making blood films in the Berlin population some years ago. This survey is still going on. We get the material within the range of other blood investigations carried through by orders

of the American Government in the American Sector of Berlin. The persons tested are of every age, sex, and social condition, and were picked without any selection out of the healthy population of Berlin. So far 16,316 individuals have been examined: among them 16 Pelgers were observed, that is to say 1 per thousand exactly. In conjunction with this the family of each proband was examined. It was possible to prove the inheritance of the anomaly in 12 families of the 16, some of them including three generations. Altogether 47 Pelger individuals have been discovered in this way.

In a paper I published together with K. Pätau[8] three years ago, we estimated the selective disadvantage of heterozygous Pelgers to be 20 percent. This estimate is running a good deal too high according to my last investigations. The Pelgers discovered belong to every age of life, from babies up to octogenerians. The proportion of Pelgers to non-Pelgers does not decrease with advancing age. We even found an especially large number of Pelgers among people 70 to 80 years old. A special sensitiveness of the Pelgers to infections has not been proved. Even though severe infections may be more detrimental to Pelgers than to non-Pelgers, there is no disadvantage under normal conditions, and the expectation of life is almost as high for Pelger as for non-Pelger, in man and in the rabbit. The full viability of the heterozygotes is in marked contrast to the lethality of the homozygotes.

Pätau and I have estimated the mutation-rate to be as high as 1:10,000. New investigations have not added further material for the estimation of the mutation rate. For that purpose we must gather material using children as probands, as it is then easier to get blood films from the preceding generation than by examining older persons. We have between 200,000 and 300,000 school children in Berlin, that would mean 200-300 Pelgers among them, if we suppose a 1 per thousand rate of Pelgers in the population. I am sorry to say, however, that it is impossible to carry out investigations of this magnitude in the quadripartite Berlin of to-day.

Summary

1. The Pelger-anomaly is an hereditary peculiarity of the nuclei of the white blood cells in man as well as in the rabbit. The nuclear segmentation—which is especially typical for the neutrophiles—is arrested. Very seldom are there more than two segments. Besides, the chromatin of the nuclei shows a strong pyknosis.

2. The Pelger-anomaly behaves as a simple Mendelian dominant with a complete penetrance; it is independent of sex.

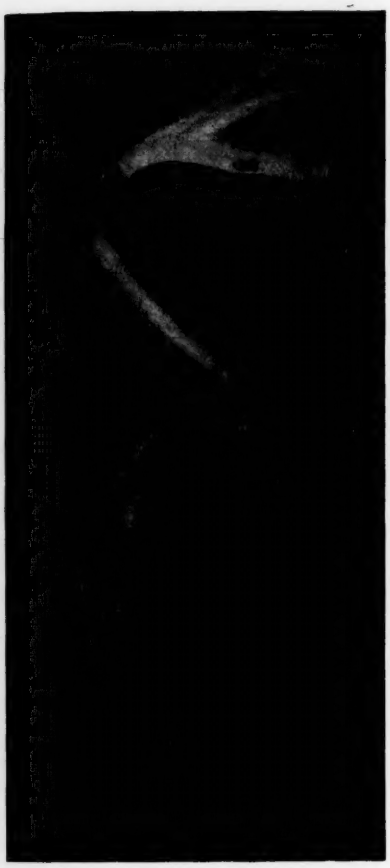
3. In the rabbit the Pelger-gene has a lethal effect in homozygous condition. Most of

TABLE I. Results of the Pelger-backcrosses.

Year	No. of litters	No. of young	Average litter size	No. of young exam.	Observed P : p	Expected P : p	Difference (± Pelger)	m	Dif/m
1940	7	46	6.57	36	20 : 16	18 : 18	+ 2	± 2.98	.67
1941	3	18	6	15	9 : 6	7.5 : 7.5	+ 1.5	± 1.90	.79
1942	18	100	5.56	82	38 : 44	41 : 41	— 3	± 4.52	.66
1943	7	39	5.57	39	17 : 22	19.5 : 19.5	— 2.5	± 3.10	.81
1944	26	130	5	125	60 : 65	62.5 : 62.5	— 2.5	± 5.59	.45
1945	1	9	9	9	4 : 5	4.5 : 4.5	— .5	± 1.49	.34
1946	9	51	5.67	38	21 : 17	19 : 19	+ 2	± 3.07	.65
1947	11	65	5.91	59	27 : 32	29.5 : 29.5	— 2.5	± 3.83	.65
1948	14	58	4.14	51	21 : 30	25.5 : 25.5	— 4.5	± 3.51	1.28
	96	516	5.38	454	217 :237	227 :227	—10	±10.64	.94

TABLE II. Results of mating heterozygous Pelger rabbits.

Year	No. of litters	No. of young	Average litter size	No. of young examined	Observed pp : Pp : PP	Expected pp : Pp : PP	% of PP born
1941	7	34	4.86	27	7 : 20 : 0	9 : 18 : 9	.0
1942	21	116	5.52	100	37 : 60 : 3	32.5 : 65 : 32.5	9.2
1943	26	133	5.12	128	40 : 82 : 6	40.5 : 81 : 40.5	14.8
1944	44	218	4.95	187	50 :127 : 10	59 :118 : 59	16.9
1945	5	25	5.0	22	11 : 11 : 0	7.5 : 15 : 7.5	.0
1946	12	48	4.0	45	15 : 26 : 4	13.5 : 27 : 13.5	29.6
1947	21	89	4.24	89	31 : 51 : 7	27.5 : 55 : 27.5	25.5
1948	28	110	3.93	103	32 : 62 : 9	31.5 : 63 : 31.5	28.6
	164	773	4.71	701	223 :439 : 39	220.5:441 :220.5	17.7



FORELEG OF SUPER-PELGER RABBIT
Figure 10

Anterior extremity of a 10½ month old rabbit showed defects of appendages reminiscent of the creeper fowl. The radius and ulna are shortened, curved and fused.

the homozygotes perish as foetuses. The homozygous Pelgers living until parturition, mostly die at this time or shortly after it. So far in only two cases (♀ and ♂) it has been possible to rear homozygous Pelger rabbits to maturity.

4. The blood picture of the homozygote is very different from that of the heterozygous Pelger rabbit. In the homozygotes the nuclear segmentation of the leucocytes is completely suppressed, the nuclei are just round. Besides the homozygotes show severe deformities of the skeletal system (phocomelia), histologically the typical picture of chondrodystrophy is found, especially in the long bones of the extremities and the ribs. Almost all homozygotes which survived parturition, perish by an emphysema of the lungs developing in consequence of a fixation of the thorax.

5. Up to now human homozygous Pelgers have not been observed. The parallelism of the symptoms of the heterozygous Pelgers in man and animals, however, leaves little doubt that the homozygotes are lethal also in man. In man and in the rabbit the viability of the heterozygotes is not essentially affected.

6. The Pelger-gene in man is more frequent than supposed. By investigations of the healthy population of Berlin 1 per mill Pelger-individuals has been found.

Literature Cited

1. Huet, G. J. *Klin. Wschr.* 11:1264-1266. II. 1932.
2. Klein, H. *Zschr. menschl. Vererbgs. u. Konstitutionsl* 29:551-620. 1949.
3. Landauer, W. *Arch. Entwicklgsmech.* 110:195-278. 1927.
4. Leitner, St. J., and P. C. Gugelot. *Acta davos.* 5:18, 10-38. 1938.
5. Nachtsheim, H. *Erbarzt.* 10:175-188. 1942.
6. ———. *Erbarzt.* 11:129-142. 1943.
7. ———. *Monhft. Veterinärmed.* 2:89-94. 1947.
8. Pätau, K., and H. Nachtsheim. *Zschr. Naturforschg.* 1:345-348. 1946.
9. Pelger, K. *Ndld. Tschr. Geneesk.* 72: 1178. IA. 1928.
10. Sar, A. van der. *Amer. Jour. Clin. Pathol.* 14:544-548. 1944.
11. Tileston, W. *Ann. int. Med.* (Amer.). 11:675-681. 1937.
12. Undritz, E. *Schweiz. med. Wschr.* 69: 1177-1186. 1939.

INHERITANCE OF BROWN AND GREEN MATURE FRUIT COLOR IN PEPPERS

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THE chocolate brown color of the ripe fruit in pepper, *Capsicum annuum** occurs commonly in Mexico and quite probably in all parts of the American tropics where this species is cultivated, according to Bukasov[3] and Bravo.[2] Several people familiar with peppers have reported its frequent cultivation in Mexico, and the author has seen small fields of it grown by Mexicans in Southern California. Little mention of it, however, appears in the literature.

Atkins and Sherrard[1] in 1915 attempted unsuccessfully to determine the nature of the brown color, which they apparently believed to be a single pigment. Recently Smith[7] reported that brown was apparently inherited as a simple recessive to red. Further, on the basis of pigment extractions and analyses made by Dr. F. P. Zscheile, it was noted that the brown color was apparently due to the presence of undecomposed chlorophyll in combination with the normal red pigments of ripe fruit. The results described below are a continuation of the study mentioned above.

Genetics and Nature of Brown Fruit Color

Initial crosses were made between normal red and brown fruited types (both obtained from Mexico), and backcrosses with the F_1 were made to each parent. The results (Table I) show the brown color to be inherited as a simple recessive to red.

The brown fruited Ac. 401 was then crossed with Oshkosh, a variety having an orange-yellow ripe fruit color which is inherited as a simple recessive to red. If a recessive gene for retention of chlorophyll were present, as believed, it would be possible to obtain, through recombination, a form having a green ripe fruit color. This combination would be genetic proof of the pigment analysis, as well as a fruit color hitherto unknown in ripe pepper fruit.

The results of this cross (Table II) gave a good 9:3:3:1 ratio, indicating that the genes for chlorophyll breakdown or retention at fruit maturity are inherited independently of the genes *R-r* for red and yellow fruit color.

Assigning the symbol *Cl* for the normal decomposition of chlorophyll at maturity and *cl* for the retention of chlorophyll, the genotypes for the four ripe fruit colors would be:

Red $= R\,Cl$
Brown $= R\,cl$
Yellow $= r\,Cl$
Green $= r\,cl$

The intensities of color in both the brown and green may be modified by other genes. In the brown fruited forms, a range from a reddish brown to deep chocolate brown has been observed, resulting from varying intensities of either contributory pigment. Although no genetic analysis has ever been attempted, the red pigment differs in intensity between varieties.

Odland and Porter[6] have shown that the intensity of green in the immature state is governed by two sets of genes G_1g_1 - G_2g_2, which produce three levels of green and one level of sulphury white.

In addition to these three levels of green, the green ripe fruit has the *r* gene governing yellow, the shade ranging from golden to orange. The cross reported above involved the variety Oshkosh, which at Davis, California, develops a definite orange yellow color when

*The use of the binominal *C. annuum* rather than *C. frutescens* is based on data, to be published soon, of the author and Charles B. Heiser, Jr.

ripe. When immature the fruit is medium green in color, while that of the brown parent, Ac. 401, is a deep green. In the eleven plants with green ripe fruit, the ripe color varied from another unattractive yellowish green to a fairly clear deep green with a somewhat olive tint. An even clearer deep green may possibly be secured by crossing varieties with deep green immature color and with a clear yellow mature color. That the yellow pigments are present but masked by chlorophyll has been frequently observed in sun-exposed fruits. On the exposed side, the chlorophyll often bleaches out, revealing the yellow pigments while the unexposed portion of the fruit remains green.

The mode of action of the *cl* gene is not known. It is quite possible, however, that the *Cl* gene controls the development of an enzyme necessary for the decomposition of chlorophyll at fruit ripening, but in the presence of the homozygous recessive *cl* gene this enzyme is not produced.

For practical breeding purposes, the double recessive green may have some value when incorporated into certain varieties of salad peppers. Not infrequently the development of red pigments in the ripening fruit renders the salad type peppers unmarketable.

Linkage Studies

In the relatively small amount of genetic investigation on the species, only one instance of linkage has been reported. Despande[4] found linkage between flower color and pigmentation on the stems.

The crosses used permitted observations on possible linkage between the *Cl-cl* genes and the genes for fruit position and pungency. Since Odland[5] found no linkage between any of the latter genes, these were not calculated.

Pungency and Fruit Color

That pungency is inherited as a single dominant gene has been shown by a number of investigators. The relatively poor 9:3:3:1 ratio observed here was due to a deficiency in the non-pungent class. Since the organoleptic method of pungency determination was used, some error is understandable when a total of 233 fruits had to be tasted. However, no linkage was evident.

Fruit Position and Color

A fair 9:3:3:1 ratio was obtained, indicating no evident linkage. The low *P* value is probably due to the deficiency of erect fruited plants rather than to any possible linkage.

TABLE I. Inheritance of ripe fruit color in crosses between red fruited (Ac. 406) and brown fruited (Ac. 401) varieties.

Cross	Generation	Red	Brown	Calculated Ratio	*P* Value
Red × Brown	F_1	19	0	—	—
Red × Brown	F_2	270	99	3:1	0.30-0.50
Red × Brown F_1 × Brown	BC	18	19	1:1	0.80-0.90
Red × Brown F_1 × Red	BC	41	—	—	—

TABLE II. Ripe fruit colors resulting from the cross of the yellow fruited variety, Oshkosh, and the brown fruited variety, Ac. 401.

Cross	Fruit color				Calculated Ratio	*P* Value
	Red	Brown	Yellow	Green		
Yellow × Brown F_1	10	0	0	0	———	———
Yellow × Brown F_2	132	46	44	11	9:3:3:1	0.50-0.70

TABLE III. Relation of pungency and fruit position to the *Cl-cl* genes in the F_2 of the cross of Oshkosh (non-pungent, upright, yellow fruit) × Ac. 401 (pungent, pendant, brown fruit).

Segregation for pungency and chlorophyll presence

	Pungent Red or Yellow	Pungent Brown or Green	Non-pungent Red or Yellow	Non-pungent Brown or Green	Calculated Ratio	*P* Value
No. observed	147	39	29	15	9:3:3:1	0.5-.10
No. expected	129.4	43.1	43.1	14.4		

Segregation for fruit position and chlorophyll presence

	Pendant Red or Yellow	Pendant Brown or Green	Erect Red or Yellow	Erect Brown or Green	Calculated Ratio	*P* Value
No. observed	137	44	29	10	9:3:3:1	.10-.20
No. expected	123.8	41.2	44.2	13.8		

Summary

The brown color of ripe pepper fruit is a result of the combination of the normal red pigments and undecomposed chlorophyll. The retention of chlorophyll in ripe pepper fruits is inherited as a simple recessive to the normal chlorophyll decomposition, and the symbols *Cl-cl* assigned for the gene pair. Crossing the brown fruited form with a yellow fruited variety, resulted in an F_1 that was red and an F_2 that segregated 9 red : 3 brown : 3 yellow : 1 green. The last color represents a ripe fruit color new for this species.

No evidence of linkage was found between the *Cl-cl* gene pair and the genes for red or yellow ripe fruit color, pungency, or fruit position.

Literature Cited

1. Atkins, W. R. G., and G. D. Sherrard. *Sci. Proc. Royal Dublin Soc.* 14:328-335. 1915.
2. Bravo, H. H. *Anal. Inst. Biol., Univ. Nac. Mexico* 5:303-321. 1934.
3. Bukasov, S. M. *Bull. Appl. Bot. Genet. and Plant Breeding, Suppl.* 47. pp. 261-273. 1930. (In Russian)
4. Despande, R. B. *Indian Jour. Agr. Sci.* 3:219-300. 1933.
5. Odland, M. L. *Minn. Agr. Expt. Sta. Tech. Bul.* 179. 1948.
6. ———— and A. M. Porter. *Proc. Amer. Soc. Hort. Sci.* 36:647-657. 1938.
7. Smith, Paul G. *Science* 107:345-346. 1948.

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THE WORLD OF LIFE

IT is refreshing to find a textbook* that carries a central theme from cover to cover —Evolution. Dr. Pauli does not divert from this theme throughout the diverse ramifications of astronomy, chemistry, physics, and biology. However, his idea of evolution, although clearly developed in Part I along astronomical lines, seems to have a restricted application. He states, "This Theory can be expressed with the greatest simplicity: The specialized creatures of today have descended by gradual change from different and usually simpler creatures of the past." Yet he devotes some space to "Cosmic evolution" and chemical and physical changes, although by definition these are excluded. This reviewer would criticize Dr. Palui's repeated assertion "the theory of evolution." He clearly defines evolution and then proceeds to devote over 600 pages to showing that the specialized creatures of today *have been* derived from simpler creatures of the past. It is not a theory but a doctrine.

In Dr. Pauli's excellent book, the orthodox arrangement of most biology texts is reversed in that paleontology preceeds the description of the modern species. This is of course consistent with the theme of evolution which is carried from cover to cover. Throughout the text, anatomy and physiology of the various phyla are only lightly stressed, while a good deal of space is devoted to a large number of individual forms within various groups.

The sequence of material seems much more logical than in many texts. For instance, Dr. Pauli postpones the discussion of the Echinoderms until after the "Highest Invertebrates —the Anthropoda," thus bringing out the fact that: "Leading toward the vertebrates are two other invertebrate groups, some of whose members are strikingly primitive compared to the highly evolved mollusks, crustaceans and insects." The summaries at the close of most of the chapters recapitulate the phase of evolution developed within the chapter, again emphasizing the central theme.

Ecological adaptations are discussed from the conventional point of view of fitness to the environment, such adaptations being brought about by mutations as the underlying cause of evolution. There is no doubt that this is a controversial subject with strong arguments on both sides. This, in spite of the fact that Dr. Pauli says "this statement (of evolution) offers no explanation of how or why such a thing should happen, but merely affirms that it did."

The book is superbly illustrated with photographic material, drawings and diagrams. The format deviates from the conventional and the double column to the page may perhaps be an aid to more rapid reading. Dr. Pauli's language is clear and understandable. It is all in all an admirable text and particularly adapted to a slightly specialized type of course in biology. As a reference book for a senior in biology it is excellent due to the fact that it ties together the entire field of biology into one continuous and coordinated whole.

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